1 The Diverse and Ubiquitous Nature of Pathogens

Diseases are pervasive throughout the biological world. They are generated by a seemingly endless 'honour' role of pathogenic organisms – fungi, oomycetes, bacteria, viruses, protozoa, nematodes, metazoa – causing symptoms ranging from the benign to the lethal in all classes of hosts – animals, plants, humans, fungi and other pathogens themselves. Many have been recognised for millennia and their impact felt through the emissaries of direct infection (e.g. the Black Death) or starvation (e.g. the Irish potato famine). However, understanding of the myriad ways in which pathogens affect the size and viability of individual host populations, of whole species, the structure of whole communities and evolutionary trajectories at all these levels has been very uneven.

Similar ecological and evolutionary forces drive the interplay of hosts and pathogens across the different biological kingdoms, and many fascinating and insightful examples can be found within each grouping (e.g. human medicine, crop pathology and veterinary science). However, the intent of this book is to provide a comprehensive view of current understanding of the part pathogens play in shaping the ecological and evolutionary dynamics of plants and the communities of which they are part. Over the last century interest in plant–pathogen interactions has developed in fits and starts in a number of distinct fields (Box 1.1). In the last few decades though, recognition of the role of plant diseases has moved from the almost exclusive preserve of agriculture and forestry to an increasing awareness of their importance as potent evolutionary forces shaping the size and structure of individual populations, the distribution of species as a whole and the nature of entire plant communities. The nature of these interactions is highly variable, reflecting differences in the longevity

BOX 1.1 Development of interest in the role of pathogens as driving forces in evolution.

Over the last century or so, major changes have occurred in perceptions regarding the role of plant pathogens in shaping ecological and evolutionary change. Starting with an understanding of the genetic basis of disease resistance (especially to rusts of cereals), there was early recognition of the disease interplay occurring at the interface between agricultural crops and surrounding wild or weedy vegetation. Indeed, barberry eradication campaigns in the Great Plains of the United States in the first few decades of the twentieth century reflected observations of early-season development of wheat stem rust in proximity to the over-wintering alternate host (barberry) – early acknowledgement of how host and pathogen life-history traits can affect the epidemiological and genetic dynamics of a system. However, the drive to develop an understanding of host–pathogen associations in agriculture was not without its mistakes. An emphasis on ecological

BI.1, FIGURE (A) Barberry eradication poster (A black and white version of this figure will appear in some formats. For the colour version, please refer to the plate section.) (Photograph by kind permission of the USDA-ARS Cereal Disease Laboratory, St Paul, Minnesota).

BOX 1.1 **(cont.)**

interactions tended to overlook the evolutionary implications of particular actions – resulting in the occasional occurrence of major unintended consequences, such as the classic 'boom-and-bust' cycles that resulted from the sequential use of single major genes for resistance.

During the mid-part of the century, theoretical population geneticists recognised the potential importance of pathogens as selective forces in evolution. Indeed, work in this area by Wright, Haldane and others (Haldane, 1949; Wright, 1951) foreshadowed by 30–40 years the importance of linking demography and genetics with space and time to obtain an evolutionary viewpoint. Moreover, their strong interest in animal and human diseases also reflected an influence (of animal and human studies) that has dominated the broader host–pathogen evolutionary story until very recently.

Simultaneously, but unlinked, Flor's classic work on gene-for-gene interactions (Flor, 1956), the work of Vavilov, Harlan and others recognising the importance of crop wild relatives as sources of disease resistance (Vavilov, 1949; Harlan, 1976; Leppik, 1976, and Person's early population-genetics models (Person, 1966) led to an increasing interest in the processes involved in host–pathogen coevolution. However, even at the beginning of the 1970s, when there was a significant increase in reductionist studies of the demography of

B1.1, FIGURE (B) Rust infected wheat in close proximity to a field-side barberry plant. (A black and white version of this figure will appear in some formats. For the colour version, please refer to the plate section.) (Photograph by kind permission of the USDA-ARS Cereal Disease Laboratory, St Paul, Minnesota).

BOX 1.1 **(cont.)**

individual plant species, interest in plant pathogens was still largely restricted to the realms of agriculture, forestry, theoretical population genetics and the conservation of plant genetic resources.

Since that time, these many separate interests and independent lines of inquiry have tended to coalesce into a more comprehensive view of the complexity of plant–pathogen associations and their ecological and evolutionary dynamics. This view recognises their role in shaping plant populations, species and communities; tackles the increasing importance of invasive and newly emerging diseases; and gives broader recognition to the fundamental importance of the influence of space and time (as manifest in the metapopulation concept and Thompson's geographic mosaic theory of coevolution [Thompson, 2005]) in driving evolutionary trajectories. Indeed, to date, plant–pathogen research has provided some of the strongest support for links between ecological and evolutionary dynamics.

of particular interactions (long-term vs recent), the epidemiological behaviour of pathogens (endemic vs epidemic) and the spatial scale of association (local vs regional).

Invasive pathogens encountering naïve hosts may change the structure of communities by 'surgically' removing individual species. For example, loss of the American chestnut, *Castanea dentata,* from West Virginian forests (Agrawal & Stephenson, 1995) or of redbay, *Persea borbonia*, from Georgia forests (Spiegel & Leege, 2013) as a consequence of the impact of *Cryphonectria parasitica* and *Raffaelea lauricola*, respectively. Invasive pathogens may also have more obvious widespread effects by destroying suites of related and unrelated species thereby transforming pre-invasion communities to a totally different state (e.g. changes induced by *Phytophthora cinnamomi* invasion of proteaceae-rich ecosystems in Western Australia; Wills, 1993). Similar effects may also result from the local *de novo* generation of novel pathogens through genetic recombination of

pre-existing ones (e.g. Ioos et al., 2006). Equally, though, invasive species may have little or no impact, essentially remaining curiosities for taxonomic aficionados, as is seen in the occurrence in the early twenty-first century of *Melampsoridium hiratsukanum* on *Alnus* sp. in northern Sweden (although damage attributable to this pathogen is increasing; L. Ericson & J. J. Burdon, personal observation), or *Puccinia malvacearum* on Malvaceous species across the globe.

Naturally occurring pathogen species show similar unpredictability in their occurrence and impact. Some are marked by high-amplitude 'boom-and-bust' epidemic cycles occurring nearly every growing season (e.g. *Melampsora* spp. occurring on *Salix* species (willow) in northern Scandinavia); while for others, high epidemic peaks are interspersed with variable numbers of years when the pathogen is virtually absent (e.g. epidemics of *M. lini* on *Linum marginale* in the Snowy Mountains of Australia; Jarosz & Burdon, 1992). In contrast to species with highly epidemic population behaviour, some pathogens show more endemic patterns of occurrence. Among those are species that induce permanent systemic infections (e.g. many floral smuts, such as *Sporisorium amphilophis* infecting *Bothrichloa macra*; García-Guzmán et al., 1996; or *Microbotryum violaceum* on *Silene dioica*; Carlsson et al., 1990); species that avoid catastrophic crashes in population size because of an ability to survive off-seasons through saprophytic growth on dead host debris (e.g. many necrotrophic pathogens of herbaceous species); and yet others that for no obvious reason always seem to be uncommon.

1.1 PLANT PATHOGENS ARE EVERYWHERE

The number of pathogens found on a given host species varies considerably, from just a few to many hundreds (e.g. >350 in *Zea mays*; Farr et al., 1989), although the number is undoubtedly influenced by the intensity of assessment (which in turn is affected by the economic importance of the host in question [Clay, 1995]). However, appropriate analysis provides strong evidence that geographic range (Strong & Levin, 1975; Miller, 2012), host size or growth form (Strong & Levin,

1979), phylogenetic relationships (Clay, 1995; Gilbert & Webb, 2007; Miller, 2012) and life-history (e.g. annual, caespitose perennial or rhizomatous perennial grasses [Clay, 1995]) all contribute to variability in numbers. Furthermore, it would appear that hosts introduced into new geographic ranges acquire a 'typical' pathogen load relatively rapidly (within the space of hundreds rather than thousands of years) through subsequent migratory 'catch-up' by its native pathogens or host range expansion by pathogens occurring on related or more distant hosts in its new home (Strong & Levin, 1975, 1979; Mitchell et al., 2010).

Despite the large numbers of pathogens associated with many individual host species, in any given geographic region only very few may cause sufficient damage as to have a selective effect, although in most associations, variable and often over-powering environmental effects make the detection of such impact hard. Pathogens that appear to be of minor importance may be being held in check by life-history constraints (e.g. microcyclic rusts in high altitude or latitude communities), prevailing environmental conditions (e.g. prolonged dry spells: Jarosz & Burdon, 1992), spatial and density constraints imposed by the host (Parratt, Numminen, & Laine, 2016), the presence of other microbiota (Bartoli et al., 2018), high levels of resistance (Laine et al., 2011) or limited seasonal growing conditions. The impact of pathogens is strongly influenced by environmental interactions. Thus for 'damping-off' diseases the probability of successful infection of the germinating seed's hypocotyl is often dependent on temperature and humidity at the soil surface; the extent of snow-blight infections of herbaceous and woody plants in the boreal zone is driven by the depth and persistence of snow pack (Olofsson et al., 2011); infections may be exacerbated by high nitrogen levels, although the particular form, nitrate or ammonium, is important (Huber & Watson, 1974); rust disease caused by *M. lini* was greatest in *Heperolinum californicum* populations growing on low calcium soils (Springer, 2009); and the three rust diseases of wheat (stem, leaf and stripe rust) each have different temperature optima for their development (McIntosh,

Wellings, & Park, 1995). Moreover, different pathogens often differ in their responsiveness to specific environmental cues (Colhoun, 1973), as can aspects of plant development, morphology and carbohydrate status that may also influence susceptibility to attack (Read, 1968). Finally, in a further complication to identifying the effects of specific pathogens, interactions between different environmental factors may also affect all aspects of disease expression.

Plants provide a wide range of distinct ecological niches for pathogens to exploit. As they progress through their life cycles, plants pass through different development phases, each of which provides opportunities for different pathogens. Each phase of growth sees the production of different host tissues that are the target of both specialist and more generalist pathogens. Thus, during germination, plants are vulnerable to relatively unspecialised 'damping-off' pathogens that attack the hypocotyls; subsequently produced leaves may be attacked by a wide range of necrotrophic and biotrophic fungal pathogens, viruses or bacteria causing spots and blotches; buds, flowers and fruits are the target of many smuts and rots; while stems and trunks are subject to vascular diseases and heart rots (Figure 1.1). In addition to this temporal diversity of potential habitats, the growth forms of different plant species show varying levels of morphological complexity, with herbs, shrubs and trees showing an increasing pattern of habitat heterogeneity that is reflected in the number of fungal pathogen species they harbour (Strong & Levin, 1979). Furthermore, the ontogenetic susceptibility of host tissue may change as it matures. For example, oak powdery mildew typically infects fresh shoots rather than mature leaves (Feau et al., 2012).

1.2 SPECIALISTS AND GENERALISTS

Plant pathogens are taxonomically a highly diverse group that includes true fungi, parasitic algae, bacteria, viruses and mycoplasma organisms. This diversity is simultaneously both a benefit to scientists in providing a broad range of experimental options and a major

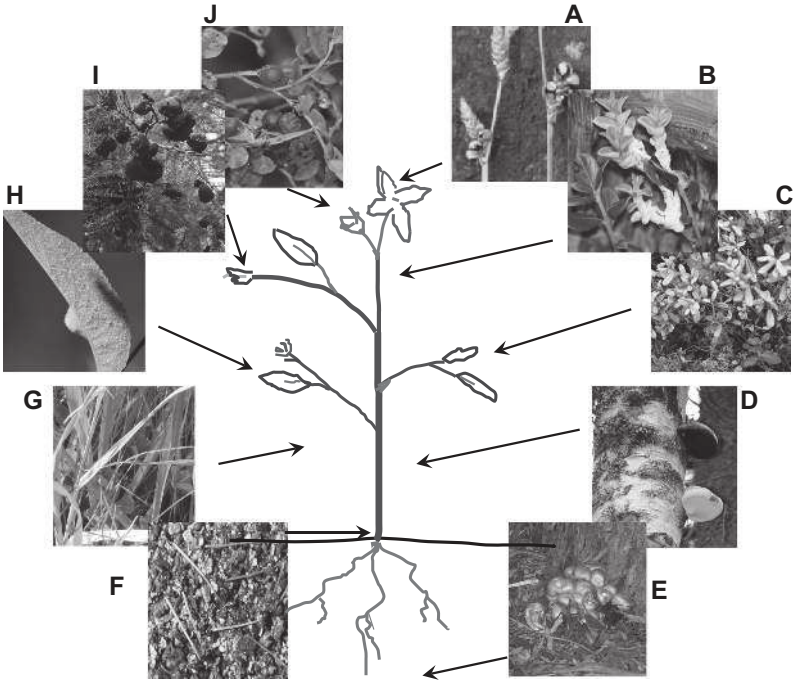

FIGURE 1.1 A stylised plant with a small selection of the range of leaf, flower, fruit, stem and
represented by *Anthracoidea fischeri* on *Carex canescens* × *mackenziei*; B: local systemic infe
on *Vaccinium vitis-idaea*; C: necrotrophic leaf diseases represented by *Eupropolella vaccinii*
represented by *Piptoporus betulinus* on *Betula pubescens*; E: root disease represented by *Armil*
damping-off represented by *Pythium* sp. on *Lepidium sativum*; G: systemic virus represented
barbata; H: biotrophic leaf diseases represented by *Melampsora larici-pentandrae* on *Salix per*
Uromycladium tepperianum on *Acacia* sp.; J: fruit diseases represented by *Lophodermium mel*
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impediment to the documentation of underlying trends in different associations and for making subsequent broad generalisations. Given that even within individual taxonomic groupings there is a large array of different host–pathogen associations, several attempts have been made to identify trait patterns that reflect ecological or evolutionarily meaningful groupings. Specificity axes (Barrett & Heil, 2012), impact effects (viability–fecundity continua; Burdon, 1991), micro- and macro-parasites (Anderson & May, 1991), epidemiological patterns (Sache & Vallavieille Pope, 1995) and dispersal patterns (Thrall & Burdon, 1997) have all been investigated as potential classificatory approaches (Table 7.1). Each of these approaches has provided a useful tool for considering patterns of pathogen behaviour without providing the depth of analysis achieved by Lockhart and her colleagues (Lockhart et al., 1996) in their study of animal diseases, which showed the over-riding importance of mode of transmission rather than host or pathogen taxonomic affiliation.

One of the most immediately important measures, though, is the extent to which a pathogen is tied to its host for nutritional support (a measure of resource reliability) and the number of host species it can successfully parasitise (a measure of its resource base). Thus, different host–pathogen associations may be placed along an interaction continuum, from obligate parasitism through various manifestations of facultative parasitism and aggressive saprophytic associations to the 'neutrality' of various forms of mutualism and endophyty. A specificity continuum roughly parallels this obligate-facultative pattern with highly obligate pathogens more frequently conforming to specialised biotrophic lifestyles tied to just a single host species (e.g. *Puccinia chondrillina* attacking *Chondrilla juncea* or *Maravalia cryptostegiae* attacking *Cryptostegia grandiflora*), in comparison to more generalist necrotrophic pathogen types with a multiplicity of different host species and the ability to grow saprophytically. Interestingly, a recent analysis (Delaye, García-Guzmán, & Heil, 2013) suggests that while biotrophy usually represents an evolutionarily stable state, the continuum between necrotrophic and

endophytic strategies is much more fluid, with species switching in both directions between these lifestyles in ecological time.

However, general patterns in host range are marked by many individual contrary examples. Application of molecular technologies provides good examples of 'cryptic' multi-species complexes that have now been shown to merit separate taxonomic status (Le Gac et al., 2007; Rouxel et al., 2013). At the same time though, even among rust fungi – perhaps the mostly widely recognised group of highly specialised pathogens – clear examples exist of individual species with very broad host ranges. Thus, the stem rust fungus *P. graminis* has a host range of at least 365 species in more than fifty genera (Anikster, 1984). Within this taxon, more than seven well-characterised sub-specific variants (formae speciales) with overlapping and/or disjoint subsets of host species have been identified, the most important of which, wheat stem rust (*P. graminis* f. sp. *tritici*), occurs naturally on at least twenty-eight different host species (Leonard & Szarbo, 2005). Similarly, a single isolate of the South American guava rust (*Austropuccinia psidii*), which invaded Australia in the early 2000s, has now been recorded on more than 350 host species in fifty-eight genera of the Myrtaceae (Carnegie & Lidbetter, 2012; Giblin & Carnegie, 2014).

1.3 THE TOLL PATHOGENS EXACT

As Burdon (1991) emphasised in classifying plant pathogens as 'killers', 'debilitators' or 'castrators', pathogens may affect the immediate viability of individuals, reduce resources available for the production of seeds and other propagules, or prevent reproduction through colonisation of various parts of the flower or stop floral initiation altogether. The extent to which pathogens have an effect on fecundity or mortality is greatly influenced by their mode of action and their relationship with their host. Thus, in the case of some seedling diseases a single pathogen individual (i.e. that growing from a single propagule) may be responsible for the immediate death of that host. Similarly, single infections resulting from conjugation of different mating-type sporidia of a floral smut may lead to castration of the